Atrial fibrillation

Atrial fibrillation (**AF** or **A-fib**) is an abnormal heart rhythm characterized by rapid and irregular beating.[1] Often it starts as brief periods of abnormal beating which become longer and possibly constant over time.[2] Most episodes have no symptoms.[3] Occasionally there may be heart palpitations, fainting, shortness of breath, or chest pain.[4] The disease increases the risk of heart failure, dementia, and stroke.[3]

Hypertension and valvular heart disease are the most common alterable risk factors for AF.[5][6] Other heartrelated risk factors include heart failure, coronary artery disease, cardiomyopathy, and congenital heart disease.[5] In the developing world valvular heart disease often occurs as a result of rheumatic fever.[7] Lung-related risk factors include COPD, obesity, and sleep apnea.[3] Other factors include excess alcohol intake, diabetes mellitus, and thyrotoxicosis.[3][7] However, half of cases are not associated with one of these risks.[3] Diagnosis is by feeling the pulse which may be confirmed using an electrocardiogram (ECG).[8] The ECG shows no P waves and an irregular ventricular rate.[8]

AF is often treated with medications to slow the heart rate to a near normal range (known as rate control) or to convert the rhythm to normal sinus rhythm (known as rhythm control).[5] Electrical cardioversion can also be used to convert AF to a normal sinus rhythm and is often used emergently if the person is unstable.[9] Ablation may prevent recurrence in some people.[10] Depending on the risk of stroke either aspirin or anti-clotting medications such as warfarin may be recommended.[3] While these medications reduce this risk, they increase rates of major bleeding.[11]

Atrial fibrillation is the most common abnormal heart rhythm.[3] In Europe and North America, as of 2014, it affects about 2% to 3% of the population.[2] This is an increase from 0.4 to 1% of the population around 2005.[12] In the developing world about 0.6% of males and 0.4% of females are affected. The percentage of people with AF increases with age with 0.14% under 50 years old, 4% between 60 and 70 years old, and 14% over 80 years old being affected.[2] A-fib and atrial flutter resulted in 112,000 deaths in 2013, up from 29,000 in 1990.[13] The first known report of an irregular pulse was by John Baptist Senac in 1749. This was first documented by ECG in 1909 by Thomas Lewis.[3]

1 Signs and symptoms

AF is usually accompanied by symptoms related to a rapid heart rate. Rapid and irregular heart rates may be perceived as palpitations or exercise intolerance and occasionally may produce anginal chest pain (if the high heart rate causes ischemia). Other possible symptoms include congestive symptoms such as shortness of breath or swelling. The arrhythmia is sometimes only identified with the onset of a stroke or a transient ischemic attack (TIA). It is not uncommon for a patient to first become aware of AF from a routine physical examination or ECG, as it often does not cause symptoms.[12]

Since most cases of AF are secondary to other medical problems, the presence of chest pain or angina, signs and symptoms of hyperthyroidism (an overactive thyroid gland) such as weight loss and diarrhea, and symptoms suggestive of lung disease can indicate an underlying cause. A history of stroke or TIA, as well as high blood pressure, diabetes, heart failure, or rheumatic fever may indicate whether someone with AF is at a higher risk of complications.[12] The risk of a blood clot forming in the left atrium, breaking off, and then traveling in the bloodstream can be assessed using the CHADS2 score or CHA2DS2-VASc score.

1.1 Rapid heart rate

Presentation is similar to other forms of rapid heart rate and may be asymptomatic.[14] Palpitations and chest discomfort are common complaints.[14] The rapid uncoordinated heart rate may result in reduced cardiac output, with the heart being unable to provide adequate blood flow and therefore oxygen delivery to the rest of the body. Common symptoms of uncontrolled atrial fibrillation may include shortness of breath,[14] shortness of breath when lying flat, dizziness, and sudden onset of shortness of breath during the night. This may progress to swelling of the lower extremities, a manifestation of congestive heart failure. Due to inadequate cardiac output,

individuals with AF may also complain of lightheadedness,[14] may feel like they are about to faint, or may actually lose consciousness.

AF can cause respiratory distress due to congestion in the lungs. By definition, the heart rate will be greater than 100 beats per minute. Blood pressure may be variable, and often difficult to measure as the beat-by-beat variability causes problems for most digital (oscillometric)

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non-invasive blood pressure monitors. For this reason, when determining heart rate in AF, direct cardiac auscultation is recommended. Low blood pressure is most concerning and a sign that immediate treatment is required. Many of the symptoms associated with uncontrolled atrial fibrillation are a manifestation of congestive heart failure due to the reduced cardiac output. Respiratory rate will be increased in the presence of respiratory distress. Pulse oximetry may confirm the presence of hypoxia related to any precipitating factors such as pneumonia. Examination of the jugular veins may reveal elevated pressure (jugular venous distention). Lung exam may reveal crackles, which are suggestive of pulmonary edema. Heart exam will reveal a rapid irregular rhythm.

2 Causes

AF is linked to several forms of cardiovascular disease, but may occur in otherwise-normal hearts. Cardiovascular factors known to be associated with the development of AF include high blood pressure, coronary artery disease, mitral stenosis (e.g., due to rheumatic heart disease or mitral valve prolapse), mitral regurgitation, left atrial enlargement hypertrophic cardiomyopathy (HCM), pericarditis, congenital heart disease, and previous heart surgery. Additionally, lung diseases (such as pneumonia, lung cancer, pulmonary embolism, and sarcoidosis) are thought to play a role in certain people. Disorders of breathing during sleep such as obstructive sleep apnea (OSA) are also associated with AF.[15] Obesity is a risk factor for AF.[16] Hyperthyroidism and subclinical hyperthyroidism are associated with AF development.[17] Caffeine consumption does not appear to be associated with AF,[18] but excessive alcohol consumption ("binge drinking" or "holiday heart syndrome") is linked to AF.[19]

2.1 Genetics

A family history of AF may increase the risk of AF. A study of more than 2,200 people with AF found that 30 percent had parents with AF.[20] Various genetic mutations may be responsible.[21][22]

Four types of genetic disorder are associated with atrial fibrillation:[23]

- Familial AF as a monogenic disease
- Familial AF presenting in the setting of another inherited cardiac disease (hypertrophic cardiomyopathy, dilated cardiomyopathy, familial amyloidosis)
- Inherited arrhythmic syndromes (congenital long QT syndrome, short QT syndrome, Brugada syndrome)

- Non-familial AF associated with genetic backgrounds (polymorphism in the ACE gene) that may predispose to atrial fibrillation

3 Pathophysiology

In AF, the normal regular electrical impulses generated by the sinoatrial node in the right atrium of the heart are overwhelmed by disorganized electrical impulses usually originating in the roots of the pulmonary veins. This leads to irregular conduction of ventricular impulses that generate the heartbeat.

3.1 Pathology

The primary pathologic change seen in atrial fibrillation is the progressive fibrosis of the atria. This fibrosis is due primarily to atrial dilation, however genetic causes and inflammation may have a cause in some individuals. Dilation of the atria can be due to almost any structural abnormality of the heart that can cause a rise in the pressure within the heart. This includes valvular heart disease (such as mitral stenosis, mitral regurgitation, and tricuspid regurgitation), hypertension, and congestive heart failure. Any inflammatory state that affects the heart can cause fibrosis of the atria. This is typically due to sarcoidosis but may also be due to autoimmune disorders that create autoantibodies against myosin heavy chains. Mutation of the *lamin AC* gene is also associated with fibrosis of the atria that can lead to atrial fibrillation.

Once dilation of the atria has occurred, this begins a chain of events that leads to the activation of the renin aldosterone angiotensin system (RAAS) and subsequent increase in matrix metalloproteinases and disintegrin, which leads to atrial remodeling and fibrosis, with loss of atrial muscle mass. This process is not immediate, and experimental studies have revealed patchy atrial fibrosis may precede the occurrence of atrial fibrillation and may progress with prolonged durations of atrial fibrillation.

Fibrosis is not limited to the muscle mass of the atria, and may occur in the sinus node (SA node) and atrioventricular node (AV node), correlating with sick sinus syndrome. Prolonged episodes of atrial fibrillation have been shown to correlate with prolongation of the sinus node recovery time,[12] suggesting that dysfunction of the SA node is progressive with prolonged episodes of atrial fibrillation.

3.2 Electrophysiology

The normal electrical conduction system of the heart allows the impulse that is generated by the sinoatrial node (SA node) of the heart to be propagated to and stimulate the myocardium (muscular layer of the heart). When

4.1 Screening

the myocardium is stimulated, it contracts. It is the ordered stimulation of the myocardium that allows efficient contraction of the heart, thereby allowing blood to be pumped to the body.

There are multiple theories about the etiology of atrial fibrillation. An important theory is that, in atrial fibrillation, the regular impulses produced by the sinus node for a normal heartbeat are overwhelmed by rapid electrical discharges produced in the atria and adjacent parts of the pulmonary veins. Sources of these disturbances are either automatic foci, often localized at one of the pulmonary veins, or a small number of localized sources in the form of either reentrant electrical spiral waves (rotors) or repetitive focal beats; these localized sources may be found in the left atrium near the pulmonary veins or in a variety of other locations through both the left or right atrium.

Because recovery of the atria from excitation is heterogeneous, the electrical waves generated by the AF sources undergo repetitive, spatially distributed breakup and fragmentation in a process known as “fibrillatory conduction”. Another theory is the multiple wavelet theory first formulated by Moe,[24] which was experimentally proven by Allessie et al.

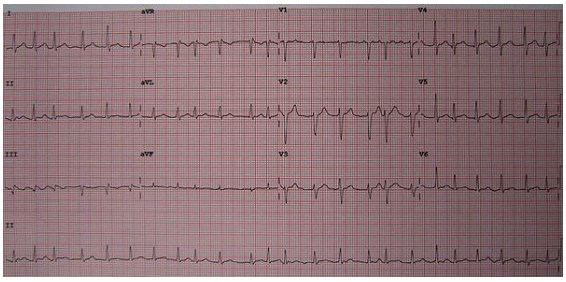
AF can be distinguished from atrial flutter (AFL), which appears as an organized electrical circuit usually in the right atrium. AFL produces characteristic saw-toothed F-waves of constant amplitude and frequency on an ECG whereas AF does not. In AFL, the discharges circulate rapidly at a rate of 300 beats per minute (bpm) around the atrium. In AF, there is no regularity of this kind, except at the sources where the local activation rate can exceed 500 bpm.

Although the electrical impulses of AF occur at a high rate, most of them do not result in a heart beat. A heart beat results when an electrical impulse from the atria passes through the atrioventricular (AV) node to the ventricles

and causes them to contract. During AF, if all of the impulses from the atria passed through the AV node, there would be severe ventricular tachycardia, resulting in severe reduction of cardiac output. This dangerous situation is prevented by the AV node since its limited conduction velocity reduces the rate at which impulses reach the ventricles during AF.[25]

4 Diagnosis

The evaluation of atrial fibrillation involves determination of the cause of the arrhythmia, and classification of the arrhythmia. Diagnostic investigation of AF typically includes a complete history and physical examination, ECG, transthoracic echocardiogram, complete blood count, and serum thyroid stimulating hormone level.[14] Depending upon given resources, afflicted individuals may benefit from an in-depth evaluation that



A 12-lead ECG showing atrial fibrillation at approximately 150 beats per minute

may include correlation of the heart rate response to exercise, exercise stress testing, chest X-ray, transesophageal echocardiography, and other studies.

If a patient presents with a sudden onset of severe symptoms, other forms of abnormal heart rhythm with high heart rate must be ruled-out, as some may be immediately life-threatening, such as ventricular tachycardia. While most patients will be placed on continuous cardiorespiratory monitoring, an ECG is essential for diagnosis. Provoking causes should be sought out. A common cause of any tachycardia is dehydration, as well as other forms of hypovolemia. Acute coronary syndrome should be ruled out. Intercurrent illness such as pneumonia may be present.

4.1 Screening

In general, screening for atrial fibrillation is not performed. Screening in those 65 years and older has been studied and been found to increase the number of cases of atrial fibrillation detected.[26]

4.2 Minimal evaluation

In general, the minimal evaluation of atrial fibrillation should be performed in all individuals with AF. The goal of this evaluation is to determine the general treatment regimen for the individual. If results of the general evaluation warrant it, further studies may then be performed.

4.2.1 History and physical examination

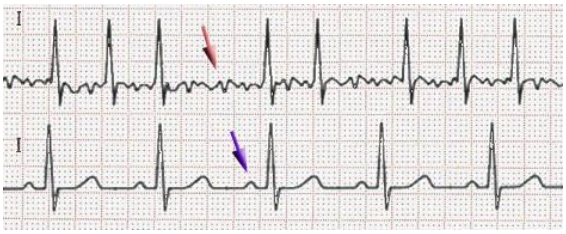
The history of the individual's atrial fibrillation episodes is probably the most important part of the evaluation. Distinctions should be made between those who are entirely asymptomatic when they are in AF (in which case the AF is found as an incidental finding on an ECG or physical examination) and those who have gross and obvious symptoms due to AF and can pinpoint whenever they go into AF or revert to sinus rhythm.

4.2.2 Routine bloodwork

While many cases of AF have no definite cause, it may be the result of various other problems. Hence, kidney function and electrolytes are routinely determined, as well as thyroid-stimulating hormone (commonly suppressed in hyperthyroidism and of relevance if amiodarone is administered for treatment) and a blood count.[12]

In acute-onset AF associated with chest pain, cardiac troponins or other markers of damage to the heart muscle may be ordered. Coagulation studies (INR/aPTT) are usually performed, as anticoagulant medication may be commenced.[12]

4.2.3 Electrocardiogram



ECG of atrial fibrillation (top) and normal sinus rhythm (bottom). The purple arrow indicates a P wave, which is lost in atrial fibrillation.

Atrial fibrillation is diagnosed on an electrocardiogram (ECG), an investigation performed routinely whenever an irregular heart beat is suspected. Characteristic findings are the absence of P waves, with disorganized electrical activity in their place, and irregular R-R intervals due to irregular conduction of impulses to the ventricles.[12] At very fast heart rates atrial fibrillation may look more regular, which may make it more difficult to separate from SVT or ventricular tachycardia.[27]

QRS complexes should be narrow, signifying that they are initiated by normal conduction of atrial electrical activity through the intraventricular conduction system. Wide QRS complexes are worrisome for ventricular tachycardia, although in cases where there is disease of the conduction system, wide complexes may be present in A-Fib with rapid ventricular response.

If paroxysmal AF is suspected but an ECG during an office visit shows only a regular rhythm, AF episodes may be detected and documented with the use of ambulatory Holter monitoring (e.g., for a day). If the episodes are too infrequent to be detected by Holter monitoring with reasonable probability, then the patient can be monitored for longer periods (e.g., a month) with an ambulatory event monitor.[12]

4.2.4 Echocardiography

In general, a non-invasive transthoracic echocardiogram (TTE) is performed in newly diagnosed AF, as well as if there is a major change in the patient's clinical state. This ultrasound-based scan of the heart may help identify valvular heart disease (which may greatly increase the risk of stroke), left and right atrial size (which indicates likelihood that AF may become permanent), left ventricular size and function, peak right ventricular pressure (pulmonary hypertension), presence of left atrial thrombus (low sensivity), presence of left ventricular hypertrophy and pericardial disease.[12]

Significant enlargement of both the left and right atria is associated with long-standing atrial fibrillation and, if noted at the initial presentation of atrial fibrillation, suggests that the atrial fibrillation is likely to be of a longer duration than the individual's symptoms.

4.3 Extended evaluation

In general, an extended evaluation is not necessary in most individuals with atrial fibrillation, and is performed only if abnormalities are noted in the limited evaluation, if a reversible cause of the atrial fibrillation is suggested, or if further evaluation may change the treatment course.

4.3.1 Chest X-ray

In general, a chest X-ray is performed only if a pulmonary cause of atrial fibrillation is suggested, or if other cardiac conditions are suspected (in particular congestive heart failure.) This may reveal an underlying problem in the lungs or the blood vessels in the chest.[12] In particular, if an underlying pneumonia is suggested, then treatment of the pneumonia may cause the atrial fibrillation to terminate on its own.

4.3.2 Transesophageal echocardiogram

A normal echocardiography (transthoracic or TTE) has a low sensitivity for identifying blood clots in the heart. If this is suspected (e.g., when planning urgent electrical cardioversion) a transesophageal echocardiogram (TEE or TOE where British spelling is used) is preferred.[12]

The TEE has much better visualization of the left atrial appendage than transthoracic echocardiography. This structure, located in the left atrium, is the place where a blood clot forms in more than 90% of cases in nonvalvular (or non-rheumatic) atrial fibrillation or flutter.[28] TEE has a high sensitivity for locating thrombi in this area[29] and can also detect sluggish bloodflow in this area that is suggestive of blood clot formation.[30]

If no blood clot is seen on TEE, the incidence of stroke,
(immediately after cardioversion is performed), is very low.

4.3.3 Ambulatory Holter monitoring

A Holter monitor is a wearable ambulatory heart monitor that continuously monitors the heart rate and heart rhythm for a short duration, typically 24 hours. In individuals with symptoms of significant shortness of breath with exertion or palpitations on a regular basis, a holter monitor may be of benefit to determine whether rapid heart rates (or unusually slow heart rates) during atrial fibrillation are the cause of the symptoms.

4.3.4 Exercise stress testing

Some individuals with atrial fibrillation do well with normal activity but develop shortness of breath with exertion. It may be unclear whether the shortness of breath is due to a blunted heart rate response to exertion caused by excessive atrioventricular node-blocking agents, a very rapid heart rate during exertion, or other underlying conditions such as chronic lung disease or coronary ischemia. An exercise stress test will evaluate the individual's heart rate response to exertion and determine if the AV node blocking agents are contributing to the symptoms.

4.4 Classification

The American College of Cardiology (ACC), American Heart Association (AHA), and the European Society of Cardiology (ESC) recommend in their guidelines the following classification system based on simplicity and clinical relevance.[12]

All people with AF are initially in the category called *first detected AF*. These patients may or may not have had previous undetected episodes. If a first detected episode stops on its own in less than 7 days and then another episode begins later on, the category changes to paroxysmal AF. Although patients in this category have episodes lasting up to 7 days, in most cases of paroxysmal AF the episodes will stop in less than 24 hours. If the episode lasts for more than 7 days, it is unlikely to stop on its own,[31] and is then known as persistent AF. In this case,

cardioversion can be used to stop the episode. If cardioversion is unsuccessful or not attempted and the episode continues for a long time (e.g., a year or more), the patient's AF is then known as permanent.

Episodes that last less than 30 seconds are not considered in this classification system. Also, this system does not apply to cases where the AF is a secondary condition that occurs in the setting of a primary condition that may be the cause of the AF.

About half of people have permanent AF while a quarter have paroxysmal and a quarter have persistent AF.[2]

In addition to the above four AF categories, which are mainly defined by episode timing and termination, the ACC/AHA/ESC guidelines describe additional AF categories in terms of other characteristics of the patient.[12]

- *Lone atrial fibrillation* (LAF) – absence of clinical or echocardiographic findings of other cardiovascular disease (including hypertension), related pulmonary disease, or cardiac abnormalities such as enlargement of the left atrium, and age under 60 years
- *Nonvalvular AF* – absence of rheumatic mitral valve disease, a prosthetic heart valve, or mitral valve repair
- *Secondary AF* – occurs in the setting of a primary condition that may be the cause of the AF, such as acute myocardial infarction, cardiac surgery, pericarditis, myocarditis, hyperthyroidism, pulmonary embolism, pneumonia, or other acute pulmonary disease

5 Management

Main article: Management of atrial fibrillation

The main goals of treatment are to prevent circulatory instability and stroke. Rate or rhythm control are used to achieve the former, whereas anticoagulation is used to decrease the risk of the latter.[32] If cardiovascularly unstable due to uncontrolled tachycardia, immediate cardioversion is indicated.[12]

5.1 Anticoagulation

Anticoagulation can be used to reduce the risk of stroke from AF. Anticoagulation is recommended in most people other than those at low risk of stroke[33] or those at high risk of bleeding.

The risk of stroke from nonvalvular AF can be estimated using the CHA_2DS_2-VASc score. For nonvalvular AF, anticoagulation is recommended if there is a score of 2 or more, not using anticoagulation may be considered if there is a score of 1, and not using anticoagulation is reasonable if there is a score of 0.[34]

Anticoagulation can be achieved through a number of means including warfarin,[35] heparin, dabigatran, rivaroxaban[36] and apixaban.[36] Aspirin is less effective in reducing the risk of stroke and may not be safer with respect to major bleeding (including intracranial bleeding) than well-managed warfarin or a non-vitamin K oral anticoagulant (NOAC).[37] A number of issues should be considered, including: cost of NOACs, risk of stroke, risk of falls, compliance, and speed of desired onset of anticoagulation.[38]

For those with non-valvular atrial fibrillation, the NOACs (rivaroxaban, dabigatran, apixaban) are not superior nor worse than warfarin in preventing non-hemorrhagic stroke and systemic embolic events.[39][40] They have a lower risk of intracranial bleeding compared to warfarin; however, dabigatran is associated with a higher risk of gastrointestinal bleeding.[39][40]

5.2 Rate versus rhythm control

There are two ways to approach atrial fibrillation using medications: rate control and rhythm control. Both methods have similar outcomes.[41] *Rate control* lowers the heart rate closer to normal, usually 60 to 100 bpm, without trying to convert to a regular rhythm. *Rhythm control* tries to restore a normal heart rhythm in a process

called cardioversion and maintains the normal rhythm with medications. Studies suggest that rhythm control is more important in the acute setting AF, whereas rate control is more important in the chronic phase.

There is no difference in risk of stroke in people having converted to a normal rhythm with anti-arrhythmic treatment compared to those with only rate control.[42] AF is associated with a reduced quality of life, and, while some studies indicate that rhythm control leads to a higher quality of life, some did not find a difference.[43]

A further study focused on rhythm control in people with AF with heart failure, based on the idea that AF increases mortality in this group. In this setting, rhythm control offered no advantage compared to rate control.[44]

In those with a fast ventricular response, intravenous magnesium significantly increases the chances of successful rate and rhythm control in the urgent setting without major side-effects.[45] A person with poor vital signs, mental status changes, preexcitation, or chest pain often will go to immediate treatment with synchronized DC cardioversion.[12] Otherwise the decision of rate control versus rhythm control using drugs is made. This is based on a number of criteria that includes whether or not symptoms persist with rate control.

5.3 Rate control

Rate control to a target heart rate of 110 bpm is recommended in most people.[46] This is achieved with medications that work by increasing the degree of block at the level of the AV node, decreasing the number of impulses that conduct into the ventricles. This can be done
with:[12][47]

- Beta blockers (preferably the "cardioselective" beta blockers such as metoprolol, atenolol, bisoprolol, nebivolol)
- Non-dihydropyridine calcium channel blockers
  (e.g., diltiazem or verapamil)

- Cardiac glycosides (e.g., digoxin) – have less use, apart from in older people who are sedentary. They are not as good as either beta blockers or calcium channel blockers.[5]

In those with chronic disease either beta blockers or calcium channel blockers are recommended.[46]

In addition to these agents, amiodarone has some AV node blocking effects (in particular when administered intravenously), and can be used in individuals when other agents are contraindicated or ineffective (particularly due to hypotension).

5.4 Cardioversion

Cardioversion is the attempt to switch an irregular heartbeat to a normal heartbeat using electrical or chemical means.[12]

- *Electrical cardioversion* involves the restoration of normal heart rhythm through the application of a DC electrical shock. Exact placement of the pads does not appear important.[48]
- *Chemical cardioversion* is performed with drugs, such as amiodarone, dronedarone,[49] procainamide, dofetilide, ibutilide, propafenone, or flecainide.

After successful cardioversion the heart may be in a stunned state, which means that there is a normal rhythm but restoration of normal atrial contraction has not yet occurred.[50]

5.5 Ablation

In young patients with little-to-no structural heart disease where rhythm control is desired and cannot be maintained by medication or cardioversion, then radiofrequency ablation or cryoablation may be attempted and is preferred over years of drug therapy.[12][51] Although radiofrequency ablation is becoming an accepted intervention in selected younger patients, there is currently a lack of evidence that ablation reduces all-cause mortality, stroke, or heart failure.[52] There are two ongoing clinical trials (CABANA [Catheter Ablation Versus Antiarrhythmic Drug Therapy for Atrial Fibrillation] and EAST [Early Therapy of Atrial Fibrillation for Stroke Prevention Trial]) that should provide new information for assessing whether AF catheter ablation is superior to more standard therapy.[53]

The Maze procedure, first performed in 1987, is an effective invasive surgical treatment that is designed to create electrical blocks or barriers in the atria of the heart, forcing electrical impulses that stimulate the heartbeat to travel down to the ventricles. The idea is to force abnormal electrical signals to move along one, uniform path to

6.2 Mitral valve

the lower chambers of the heart (ventricles), thus restoring the normal heart rhythm.[54]

5.6 Following surgery

AF often occurs after cardiac surgery and is usually self-limiting. It is strongly associated with age, pre-operative hypertension, and the number of vessels grafted. Measures should be taken to control hypertension pre-operatively to reduce the risk of AF. Also, people with a higher risk of AF, e.g., people with preoperative hypertension, more than 3 vessels grafted, or greater than 70 years of age, should be considered for prophylactic treatment. Postoperative pericardial effusion is also suspected to be the cause of atrial fibrillation. Prophylaxis may include prophylactic post-operative rate and rhythm management. Some authors perform posterior pericardiotomy to reduce the incidence of postoperative AF.[55] When AF occurs, management should primarily be rate and rhythm control. However, cardioversion may be employed if the person is haemodynamically unstable, highly symptomatic, or persists for 6 weeks after discharge. In persistent cases anticoagulation should be used.

6 Prognosis

6.1 Thromboembolism

See also: CHADS score

6.1.1 Prediction of embolism

Determining the risk of an embolism causing a stroke is important for guiding the use of anticoagulants. The most accurate clinical prediction rules are:[56]

- CHADS2
- CHA2DS2-VASc

Both the CHADS2 and the CHA2DS2-VASc score predict future stroke risk in people with a-fib with CHA2DS2-VASc]] being more accurate. Some that had a CHADS2 score of 0 had a CHA2DS2-VASc score of 3, with a 3.2% annual risk of stroke. Thus a CHA2DS2VASc score of 0 is considered very low risk.[57]

6.1.2 Mechanism of thrombus formation

In atrial fibrillation, the lack of an organized atrial contraction can result in some stagnant blood in the left atrium (LA) or left atrial appendage (LAA). This lack of movement of blood can lead to thrombus formation (blood clotting). If the clot becomes mobile and is carried away by the blood circulation, it is called an embolus. An embolus proceeds through smaller and smaller arteries until it plugs one of them and prevents blood from flowing through the artery. This process results in end organ damage due to loss of nutrients, oxygen, and removal of cellular waste products. Emboli in the brain may result in an ischemic stroke or a transient ischemic attack (TIA).

More than 90% of cases of thrombi associated with non-valvular atrial fibrillation evolve in the left atrial appendage.[28] However, the LAA lies in close relation to the free wall of the left ventricle and thus the LAA's emptying and filling, which determines its degree of blood stagnation, may be helped by the motion of the wall of the left ventricle, if there is good ventricular function.[58]

If the LA is enlarged, there is an increased risk of thrombi that originate in the LA. Moderate to severe, non-rheumatic, mitral regurgitation (MR) reduces this risk of stroke.[59] This risk reduction may be due to a beneficial swirling effect of the MR blood flow into the LA.[60]

6.2 Mitral valve

Atrial fibrillation and a corresponding enlargement of the left atrium may cause an increase in size of the mitral valve annulus.[61]

With a sinus rhythm, the mitral annulus undergoes dynamic changes during the cardiac cycle. For example, at the end of diastole the annular area is smaller than at the end of systole. A possible reason for this dynamic size difference is that the coordinated contraction of the left atrium acts like a sphincter about the mitral annulus and reduces its size. This may be important for mitral valve competence so that it does not leak when the left ventricle pumps blood. However, when the left atrium fibrillates, this sphincter action is not possible and may contribute to, or result in, mitral regurgitation in some cases.[61]

7 Epidemiology

Atrial fibrillation is the most common arrhythmia.[12] In Europe and North America as of 2014 it affects about 2% to 3% of the population.[2] This is an increase from 0.4 to 1% of the population around 2005.[12] In the developing world rates are about 0.6% for males and 0.4% for females.[2]

It also accounts for one-third of hospital admissions for cardiac rhythm disturbances,[12] and the rate of admissions for AF has risen in recent years.[62] Strokes from AF account for 6–24% of all ischemic strokes.[63] After a transient ischemic attack or stroke about 11% are found to have a new diagnosis of atrial fibrillation.[64] Between 3 and 11% of those with AF have structurally normal hearts.[65] Approximately 2.2 million individuals in the United States and 4.5 million in the European Union have AF.[12]

The number of new cases each year of atrial fibrillation increases with age. In individuals over the age of 80 it affects about 8%.[12] In developed countries, the number of patients with atrial fibrillation is likely to increase during the next 50 years, owing to the growing proportion of elderly individuals.[66]

8 History

Because the diagnosis of atrial fibrillation requires measurement of the electrical activity of the heart, atrial fibrillation was not truly described until 1874, when Edmé Félix Alfred Vulpian observed the irregular atrial electrical behavior that he termed *"fremissement fibrillaire"* in dog hearts.[67] In the mid-eighteenth century, JeanBaptiste de Sénac made note of dilated, irritated atria in people with mitral stenosis.[68] The irregular pulse associated with AF was first recorded in 1876 by Carl Wilhelm Hermann Nothnagel and termed *"delirium cordis"*,

stating that "[I]n this form of arrhythmia the heartbeats follow each other in complete irregularity. At the same time, the height and tension of the individual pulse waves are continuously changing".[69] Correlation of delirium cordis with the loss of atrial contraction as reflected in the loss of *a waves* in the jugular venous pulse was made by Sir James MacKenzie in 1904.[70] Willem Einthoven published the first ECG showing AF in 1906.[71] The connection between the anatomic and electrical manifestations of AF and the irregular pulse of delirium cordis was made in 1909 by Carl Julius Rothberger, Heinrich Winterberg, and Sir Thomas Lewis.[72][73][74]

9 See also

Ventricular fibrillation